

**THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY**

**CHENNAI - 600032**



**A STUDY ON BRAIN ABSCESS**

Dissertation submitted in partial fulfilment  
of the requirements of

**M.Ch BRANCH II NEUROSURGERY (3 YEARS)**

**EXAMINATIONS – AUGUST 2013**

INSTITUTE OF NEUROLOGY

MADRAS MEDICAL COLLEGE &

RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL

CHENNAI – 600 003

## **CERTIFICATE**

This is to certify that this dissertation entitled “A STUDY ON BRAIN ABSCESS” submitted by Dr. T. Suresh Babu, appearing for M.Ch (Neurosurgery) degree examination in August 2013 is an original bonafide record of work done from August 2010 to February 2013 by him under my guidance and supervision in partial fulfilment of requirement of the Tamil Nadu Dr. M. G. R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M. G. R. Medical University, Chennai, Tamil Nadu, India.

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## **DECLARATION**

I, Dr. T. Suresh Babu, solemnly declare that this dissertation “A STUDY ON BRAIN ABSCESS” was done by me at the Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of the Professor of Neurosurgery, Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3, between 2010 and 2013.

This dissertation is submitted to the Tamil Nadu Dr. M. G. R. Medical University, Chennai-600032 in partial fulfilment of the University requirements for the award of the degree of M.Ch. Neurosurgery.

Place : Chennai

Date : 25-03-13

(T.Suresh Babu)

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## **INTRODUCTION**

## INTRODUCTION

Brain abscess is defined as a focal, intra cerebral infection that begins as a localized area of cerebritis and develops into a collection of pus surrounded by a well-vascularized capsule.

Although rare in developed countries brain abscess still remains a significant health care problem in developing countries. Historically brain abscess was mentioned by Hippocrates in 460 BC<sup>45</sup> and he himself has described the association of brain abscess with ear infection.

The Brain is well protected from infections by a thick skull vault, tough duramater and important barriers like bloodbrain barrier and blood cerebrospinal fluid (csf) barrier. A breach in any of these barriers leads to entry of microorganisms into the brain initiating a suppurative inflammation culminating in brain abscess. The inciting organism can be introduced from outside by trauma or endogenously from infection in a contiguous site eg. ear or occasionally blood borne, heart disease and systemic infection. Although the portal of entry of the organism is commonly identified, the organism remains obscure in 10 -37% of the patients.

In the recent decades the addition of Computed tomography (CT) and Magnetic Resonance Imaging (MRI) to the diagnostic



armamentarium has facilitated early identification and thereby prompt institution of therapy in brain abscess. Improved microbiological techniques have increased the appreciation of the bacteriological spectrum of brain abscess especially anaerobes. The advanced surgical techniques viz. stereotactic guided aspiration, real-time ultrasound imaging has caused a paradigm shift in the management and outcome of brain abscess.

Despite these advancements brain abscess remains a serious infection with a mortality rate of 5-15% which escalates to as high as 80% in case of rupture.

Hence a study is conducted to observe and analyse the clinical profile, radiological features, management and outcome of patients admitted in our institute with brain abscess over a period of three years.

## **AIM OF THE STUDY**

### **AIM OF THE STUDY**

The aim is to study the epidemiology, clinical profile, management and outcome in patients with brain abscess.

## **REVIEW OF LITERATURE**

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Geographic location and living standards play a major role in the incidence of brain abscess. This is well established by the more number of cases in developing countries where the sanitation and living conditions are poor. In our sub-continent the incidence of brain abscess is 8% of intra cranial space occupying lesions<sup>1</sup>. In developed countries like UK the incidence is 1-2%.<sup>42</sup> The tropical location and the climate of our country favour organism growth than western world.

### **Aetiopathogenesis**

Brain abscess develops in association with a contiguous supportive focus, following cranial trauma, haematogenous spread from distant focus, cryptogenic<sup>2</sup> origin and due to miscellaneous causes.

### **Contiguous suppurative focus**

Para nasal sinus, middle ear and mastoid infection are the most common source of brain abscess<sup>5, 8</sup>. This occurs as a result of direct extension from osteitis or osteomyelitis, spread via diploic or emissary veins or local spread by lymphatics.

Usually the osteomyelitis is prevented from entering the brain by the tough texture of the duramater. This leads to epidural abscess formation and granulation tissue covers the outer surface of the Dura. The areas where the blood vessels traverse the dura infection spreads to the brain causing abscess. The brain is adherent to this patchy inflamed dura<sup>49</sup> which is described as stalk of abscess. It is through this stalk the abscess contents are discharged to the exterior through the hole in the dura and the bone.

### **Otogenic abscess**

In our country middle ear and mastoid infection are the most common causes of brain abscess (40-63%)<sup>4, 5</sup>. These abscesses are usually solitary and develop in the inferior part of ipsilateral temporal lobe. Mastoiditis causes infection in the ipsilateral cerebellar hemispheres. Cholesteatomas complicating csom and mastoiditis increase the risk of intra cranial spread.

**Para nasal sinus infection** leading to brain abscess is on the decline due to early diagnosis and treatment. Frontal, ethmoidal and maxillary sinus infection cause frontal lobe abscess. Sphenoidal sinusitis causes abscess in the temporal lobe or in the pituitary fossa.

### **Periodontal infection**

Infection of the molar teeth spreads between the muscles of mastication along the fascial planes to the skull causing frontal lobe abscess.

### **Post traumatic**

The incidence of brain abscess in penetrating open head injuries is 3-17%<sup>18, 39, 41</sup>. These are caused by the retained contaminated bone fragments foreign bodies, hair etc. Post traumatic abscess develops after quite a long interval after the primary injury.

### **Post-craniotomy**

Post craniotomy patients can also develop brain abscess by the organisms which may get introduced at the time of surgery, staphylococcus being the commonest organism in such cases<sup>3</sup>.

### **Metastatic abscess**

Inspite of the inherent resistance of the BBB to infection the transient bacteraemia develops into brain abscess. These metastatic abscess occur through haematogenous spread of micro organisms from other parts of the body<sup>16, 44</sup>.

Primary foci usually include skin pustules, pulmonary infections (like empyema, bronchiectasis, pneumonia) acute diverticulitis, osteomyelitis, dental abscess, infective endocarditis etc. These abscesses are difficult to differentiate from metastatic neoplasms.

Metastatic abscess are multiple and usually occurs in the corticomedullary junction where the blood flow is sparse. They occur more commonly in the MCA territory in the frontal and parietal lobes. Less commonly it occurs in thalamus, brain stem and cerebellum etc.

**Cyanotic congenital heart diseases** predispose to brain abscess because the pathogens bypass the pulmonary filtration process and enter the brain<sup>6, 7,21,25,40</sup>. Further Right to left shunts cause hypoxia which leads to polycythemia and hyperviscosity of blood and decreased perfusion causing microinfarcts. These microinfarct areas provide a fertile ground for brain abscess. Among the cardiac defects tetralogy of fallot is the commonest cause.



## Stages of Brain Abscess<sup>10, 11</sup>

The pathological stages of brain abscess are as follows.

Stage	Histologic characteristics (days shown are general estimates)	Resistance to aspirating needle
1	<u>early cerebritis</u> : (days 1-3) early infection & inflammation, poorly demarcated from surrounding brain, toxic changes in neurons, perivascular infiltrates	intermediate resistance
2	<u>late cerebritis</u> : (days 4-9) reticular matrix (collagen precursor) & developing necrotic center	no resistance
3	<u>early capsule</u> : (days 10-13) neovascularity, necrotic center, reticular network surrounds (less well developed along side facing ventricles)	no resistance
4	<u>late capsule</u> : (> day 14) collagen capsule, necrotic center, gliosis around capsule	firm resistance, "pop" on entering

## Microbiological Aspects

Numerous infectious agents have been reported to cause brain abscess. The most common bacteria are Streptococcus (aerobic, anaerobic, micro aerophilic). Staphylococcus aureus is the most common organism in brain abscess that develops following trauma, post-craniotomy and infective endocarditis<sup>4</sup>. The microorganisms in the brain abscess depend on the predisposing factors. The negative cultures have ranged from 0-47%<sup>5</sup>. This may be due to the previous use of anti microbial therapy given empirically. Mycobacterium tuberculosis and

non tuberculous mycobacteria are increasingly seen in the recent times due to the emergence of HIV.

Fungal brain abscess are commonly seen in the individuals who are on corticosteroid therapy, broad spectrum anti microbial therapy and immunosuppressive agents like transplant recipients etc.

### **Clinical Presentation<sup>8</sup>**

Majority of brain abscess occurs in the first two decades of life with a unexplained predilection for males. The clinical features depend on the size, number of lesions, virulence of organisms, host response, the brain area affected and the severity of brain oedema.

Anyhow no particular set of findings is pathognomonic of brain abscess. Symptomatically brain abscess are indistinguishable from any other brain SOL but abscess are acute and rapidly progressive in their presentation. Classical triad of fever, headache and focal neurological deficit are seen in 50% of cases<sup>31, 34, 35, 43</sup>.

Headache is the commonest symptom which may get worsened by the onset of meningitis. In immunocompromised individuals the clinical features can be masked by the reduced inflammatory response.

Nocardia brain abscess patients can have pulmonary, skin or muscle lesions. Aspergillosis brain abscess patients manifest signs of stroke syndrome. This may be a result ischaemia or intra cerebral haemorrhage.

## **Radiology**

Plain X-Ray skull, pneumoencephalography and ventriculography, were used in olden days for the evaluation of brain abscess. CT and MRI revolutionized neuroimaging and improved the diagnosis and management of brain abscess. X-Ray of skull is often normal in patients with brain abscess, but features of raised intracranial tension, pneumocephalus, mastoiditis and sinusitis can be seen.

After the advent of CT-scan, angiography, ventriculography, pneumoencephalography and radionuclide brain scanning became obsolete. CT scan hastens the diagnosis, localization and staging of brain abscess<sup>46</sup>. Features of raised intracranial tension, hydrocephalus, and subdural empyema can be identified. In plain CT the abscess appears as isodense or slightly denser than the surrounding brain tissue.

In contrast enhanced CT the abscess appears as a smooth thin walled and hypodense areas both in the centre and surrounding regions. It also helps to study the resolution of abscess on follow up.

## **MRI**

It is the diagnostic procedure of choice for abscess. Even abscesses in early cerebritis stage can be identified with accuracy with MRI<sup>22</sup>. MRI gives better anatomical details in various planes and is more useful in posterior fossa and satellite lesions. In T1 weighted images, the central necrotic area is hypointense and the surrounding oedema is less hypointense whereas the capsule is iso to mild hyperintense. On T2 weighted images the central core is iso to hyperintense, surrounding oedema is hyperintense and capsule appears as a hypointense rim.

## **MRS**

Magnetic resonance spectroscopy differentiates brain abscess from other cystic intracranial space occupying lesions. Magnetisation transfer MRI differentiates tuberculous abscess from other abscess. The presence of aminoacid is marker of pyogenic abscess and acetate indicates presence of anaerobic bacteria<sup>26, 32, 38</sup>.

## **DWI**

In diffusion weighted images abscess fluid shows as high signal intensity with low apparent diffusion coefficient. Stereotactic MRI and CT guided aspiration facilitate access to any critical, deep area of CNS and aid in the microbiological diagnosis.

## **Management**

Multidisciplinary approach involving the neurosurgeon, neuroradiologist and infectious disease specialist will help in the management of brain abscess. Every case should be individualized and managed on its own merit. Patient's age, neurological status, number of abscess, location, stage of the abscess, etc plays a major role in following either conservative or surgical approach.

### **Conservative Management** <sup>17, 23, 28,29</sup>

This is followed for abscess in the early cerebritis stage, lesion less than 3cm with no features of raised intracranial pressure (ICP). Bleeding diathesis patients and those who are not fit for any surgical procedures are treated conservatively.

A combination of vancomycin, metronidazole and a third or fourth generation cephalosporins can be used empirically. This combination significantly lowered the mortality and morbidity due to brain abscess. Early cerebritis and small abscess respond well to conservative management.

The efficacy of antibiotics depend on various factors - whether the drug is bactericidal or bacterostatic, its ability to cross CSF, blood brain

barrier (BBB), host response to infection and the drug concentration in the abscess.<sup>17,23,28</sup>

## **Surgical Management**

Indications for surgery includes

1. Deterioration in general condition of patients managed conservatively.
2. Any mass effect
3. Neurological deficit
4. Multiple lesions in accessible locations
5. Doubtful cases
6. Resistant organisms
7. Multi loculated abscess

Surgical procedures like drainage, aspiration and excision have been described but drainage is seldom used nowadays. In choosing between aspiration and excision factors like surgical, morbidity, cure rate and neurological sequale should be considered. Stereotactic and real time guided USG plays a major role in aspiration.<sup>13</sup>

Advantages of aspiration are

1. Rapid and safe procedure
2. Safer in eloquent and deep seated areas
3. Can be done with stereotaxy and real time USG
4. Can be done under local anaesthesia
5. Less invasive
6. Stereotactic aspiration can be combined with antibiotics and hyperbaric oxygen therapy<sup>13, 28</sup>.

## **Excision**

Craniotomy and excision is of two types, primary and secondary. Primary excision is indicated in cases of post traumatic abscess during which the contaminated bone fragment, foreign bodies etc are removed

Secondary excision is usually done if there is no reduction in the size of abscess and the quantity of pus, even after repeated aspiration (multiloculated abscess). Secondary excision is easier because of the reduction in surrounding oedema. Posterior fossa abscess obstructs CSF pathway causing hydrocephalus where V-P shunt is useful<sup>25, 36</sup>.

## **MATERIALS AND METHODS**



## **MATERIALS AND METHODS**

All patients admitted in the Institute of Neurology, Rajiv Gandhi Government General Hospital & Madras Medical College, Chennai, between August 2010 and February 2013, with brain abscess was included in this study.

### **Inclusion criteria**

All patients who have been diagnosed as having brain abscess were **included** in the study group.

On admission patient's clinical profile such as age, sex, presenting symptoms complete neurological examination, conscious level and signs were recorded. All patients were subjected to CT scan brain plain study. In CT of patients having brain abscess the following features were noted – location, number of abscess, loculation, associated hydrocephalus.

Patients were either managed conservatively or by surgery. The specimen obtained by surgical means was subjected to microbiological study. All the data were entered in a proforma as shown in the appendix. Outcome analysis was done at the time of discharge.

## **OBSERVATION AND RESULTS**

## OBSERVATION

### 1. Age distribution

AGE	NUMBER OF PATIENTS
Less than 1 year	13
1 to 12 years	8
13 to 20 years	12
21 to 30 years	11
31 to 40 years	8
41 to 50 years	3
51 to 60 years	3
61 to 70 years	2
<b>Total</b>	<b>60</b>

Table 1: Age distribution

Age distribution of the total 60 patients is shown in table – 1. Majority of patients belonged to first decade, followed by second and third decade.

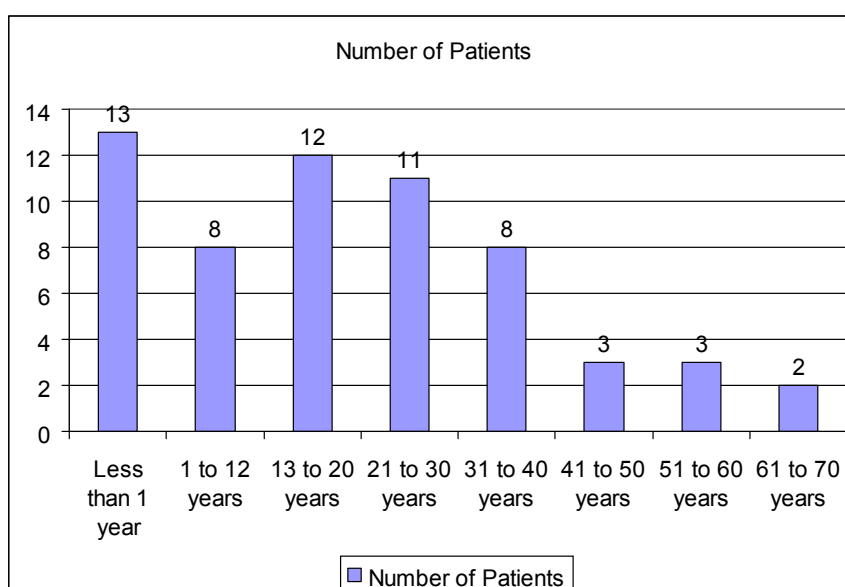


Chart 1: Age distribution

## 2. Sex distribution

SEX	FREQUENCY
FEMALE	16
MALE	44
Total	60

Table 2: Sex distribution

Of the total 60 patients who had brain abscess, 44 were male and 16 were female.

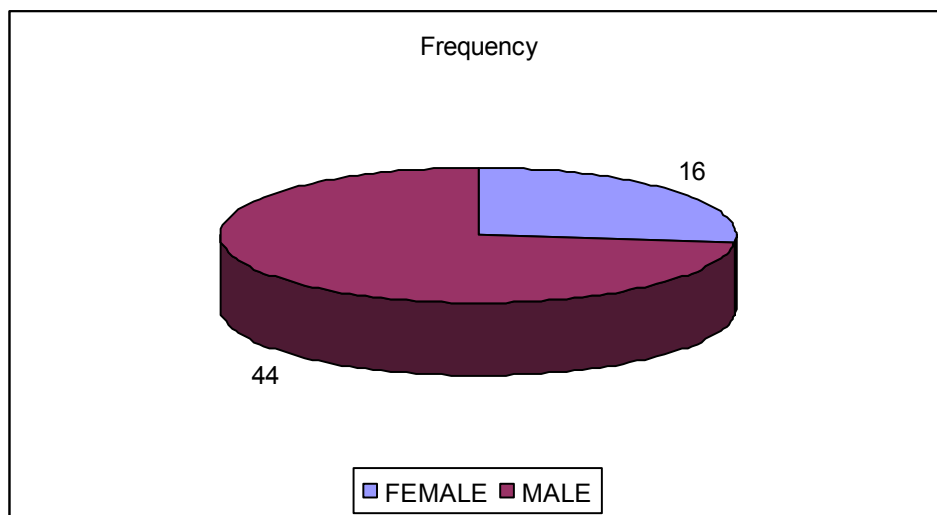


Chart 2: Sex distribution

The above chart depicts sex distribution in the study.

### 3a. Predisposing factors – Trauma

TRAUMA	FREQUENCY
NO HISTORY OF TRAUMA	54
HISTORY OF TRAUMA	6
Total	60

Table 3: Predisposing factors – Trauma

6 of the patients had history of trauma as a predisposing factor whereas majority of the abscess were non traumatic.

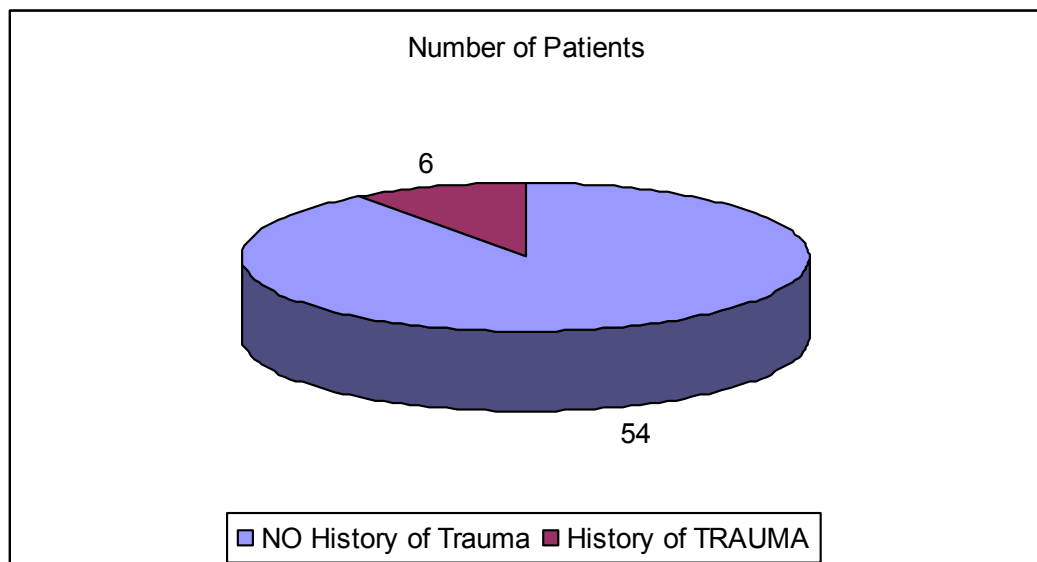


Chart 3: Predisposing factors – Trauma

### 3b. Predisposing factors - Congenital heart disease

CONGENITAL HEART DISEASE	FREQUENCY
TGAVSD	1
TOF	3
TOF + PULMONARY ATRESIA	1
Total	5

Table 4: Predisposing factors – Congenital heart disease

5 out of 60 patients had congenital heart disease which predisposed to brain abscess.

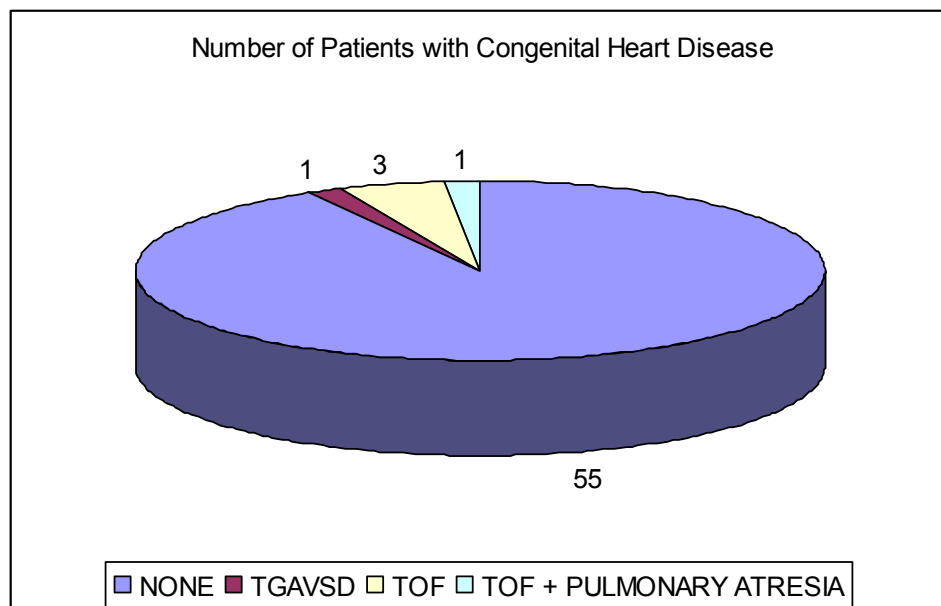


Chart 4: Predisposing factors – Congenital heart disease

### 3c. Predisposing factors – Lung pathology

LUNG	FREQUENCY
BRONCHIECTASIS	1
PNEUMONIA	1
PNEUMONIA-SLE	1
Total	3

Table 5: Predisposing factors – Lung pathology

3 out of 60 patients had lung pathology which predisposed to brain abscess.

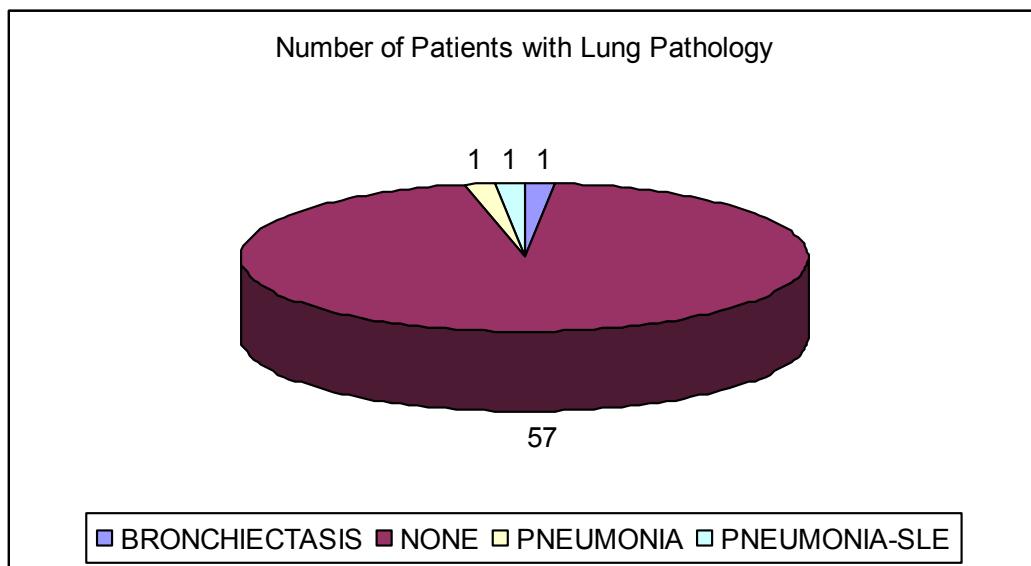


Chart 5: Predisposing factors – Lung pathology

### 3d. Predisposing factors – ENT focus

ENT	FREQUENCY
FRONTAL SINUSITIS	1
LEFT CSOM	6
RIGHT CSOM	3
Total	10

Table 6: Predisposing factors – ENT focus

10 of the patients had ENT focus as a predisposing factor for brain abscess.

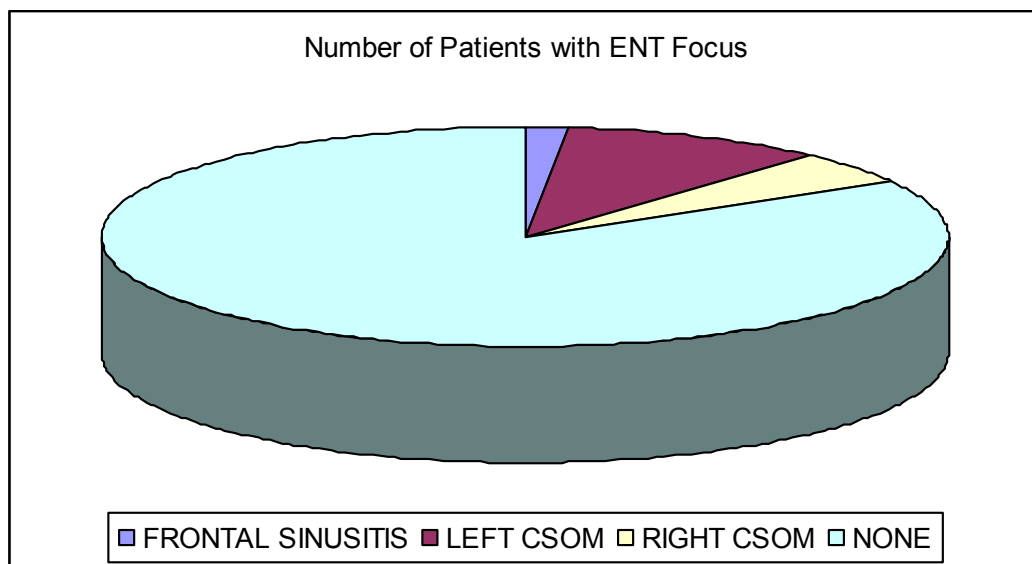


Chart 6: Predisposing factors – ENT focus



### 3e. Predisposing factors – Other Causes

OTHER ASSOCIATED DISEASES	FREQUENCY
HIV	2
ORBITAL CELLULITIS	1
PRE TERM / SEPSIS	4
SEPSIS	1
THIGH ABSCESS ,CGD	1
<b>Total</b>	<b>9</b>

Table 7: Predisposing factors – Other Causes

9 patients had other causes which predisposed to brain abscess. The other causes being HIV, orbital cellulitis, pre term sepsis, thigh abscess and Chronic Granulomatous Disease (CGD)

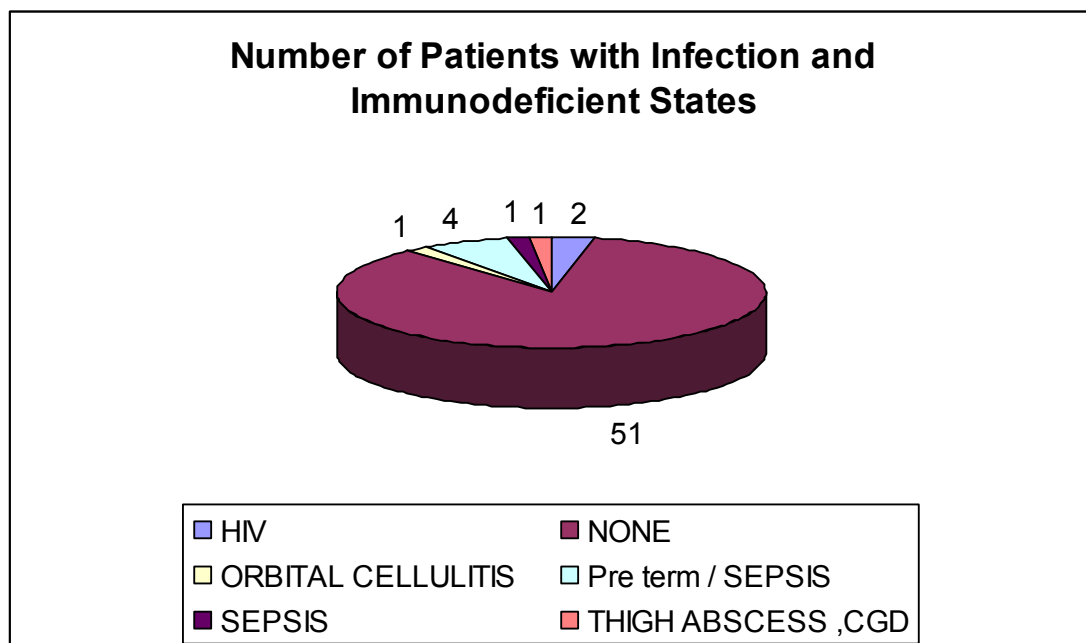


Chart 7: Predisposing factors – Other Causes

### 3f. Predisposing factors – Consolidated

PREDISPOSING CAUSE	NO. OF PATIENTS AFFECTED
FRONTAL SINUSITIS	1
LEFT CSOM	6
RIGHT CSOM	3
TGAVSD	1
TOF	3
TOF + PULMONARY ATRESIA	1
BRONCHIECTASIS	1
PNEUMONIA	1
PNEUMONIA-SLE	1
HIV	2
ORBITAL CELLULITIS	1
PRE TERM / SEPSIS	4
SEPSIS	1
THIGH ABSCESS ,CGD	1
TRAUMA	6
NONE	27
<b>TOTAL</b>	<b>60</b>

This table shows the pre disposing factors for brain abscess

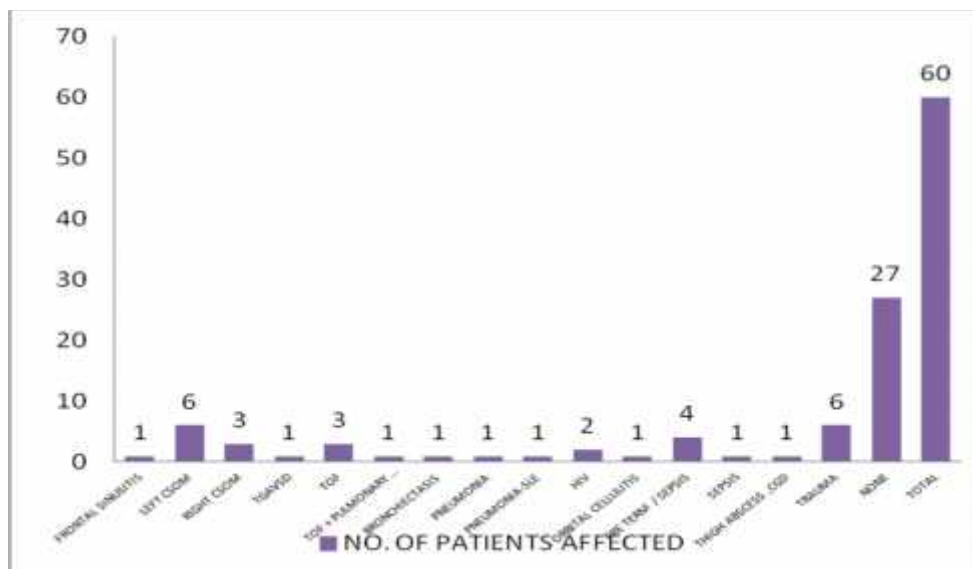


Chart8: Predisposing factors – Causes consolidated

#### 4a. Clinical manifestations – Fever

FEVER	NUMBER OF PATIENTS
PATIENTS WITH FEVER	52
PATIENTS WITH NO FEVER	8
Total	60

Table 9: Clinical manifestation – Fever

Majority of the patients with brain abscess had fever

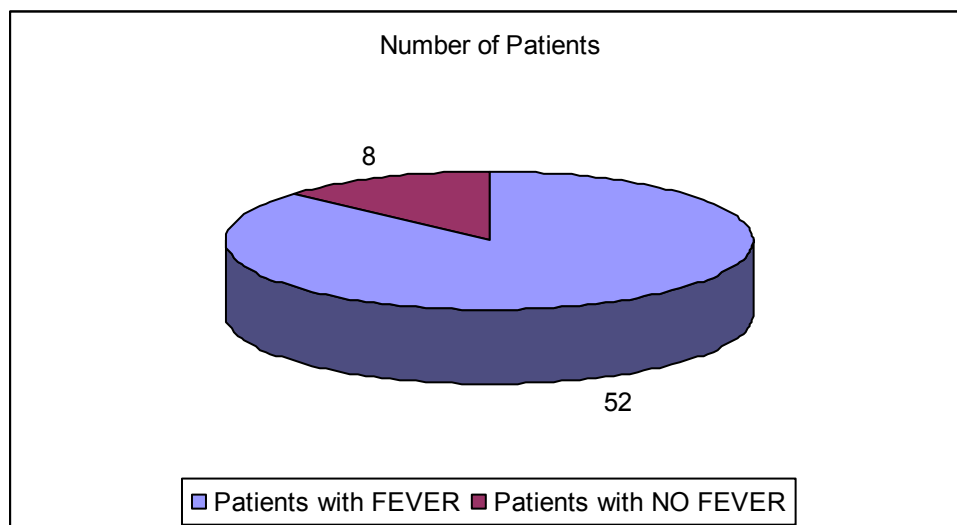


Chart 9: Clinical manifestation – Fever

#### 4b. Clinical manifestations – Headache

HEADACHE	NUMBER OF PATIENTS
PATIENTS WITH HEADACHE	38
PATIENTS WITH NO HEADACHE	22
Total	60

Table 10: Clinical manifestation – Headache

More than 50% of the patients with brain abscess had headache

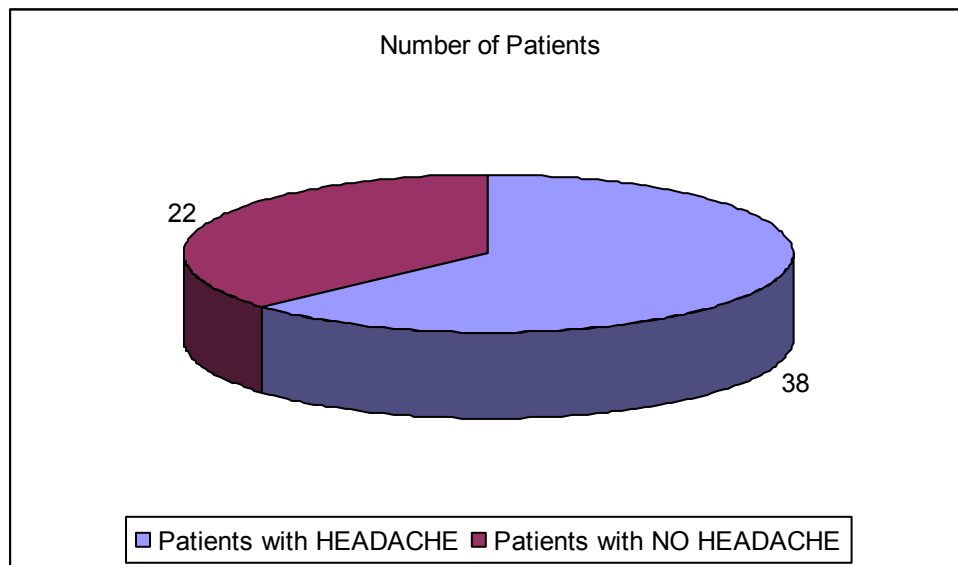


Chart 10: Clinical manifestation – Headache

#### 4c. Clinical manifestations - Seizures

SEIZURES	NUMBER OF PATIENTS
PATIENTS WITH NO SEIZURES	36
PATIENTS WITH SEIZURES	24
Total	60

Table 11: Clinical manifestation – Seizures

24 of the 60 patients with brain abscess had seizure at presentation

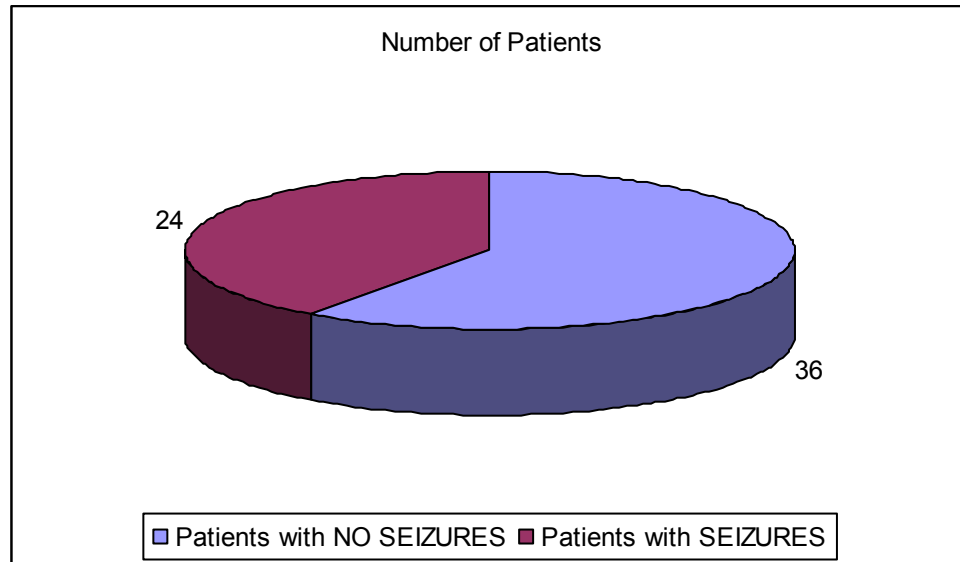


Chart 11: Clinical manifestation – Seizures

#### 4d. Clinical manifestations - Vomiting

VOMITING	NUMBER OF PATIENTS
PATIENTS WITH NO VOMITING	24
PATIENTS WITH VOMITING	36
<b>TOTAL</b>	<b>60</b>

Table 12: Clinical manifestation – Vomiting

36 of the 60 patients with brain abscess had vomiting at presentation

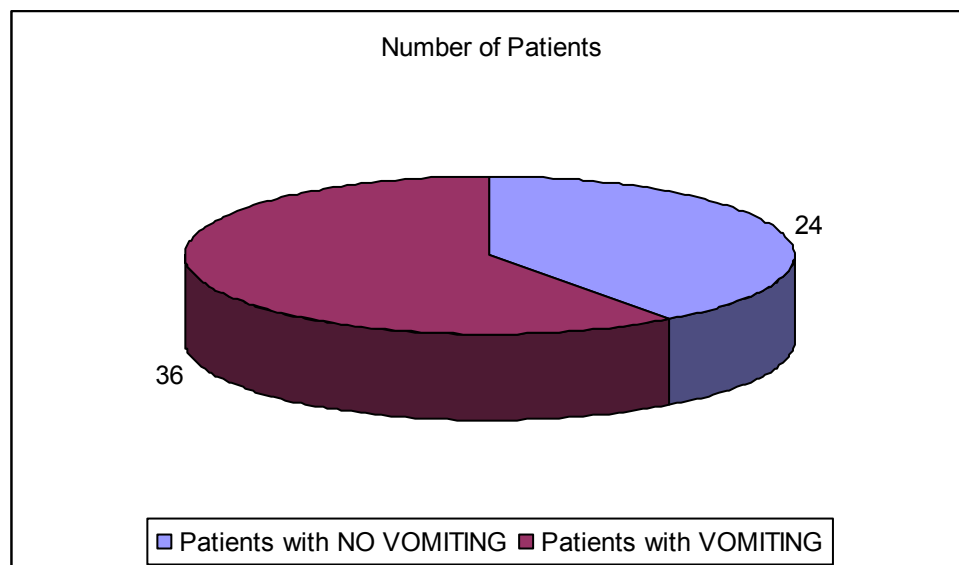


Chart 12: Clinical manifestation – Vomiting

#### 4e. Clinical manifestations – Focal neurological deficit

FOCAL NEUROLOGICAL DEFICITS	FREQUENCY
L HEMIPARESIS	6
R HEMIPARESIS	5
Total	11

Table 13: Clinical manifestation – Focal neurological deficit

11 of the 60 patients with brain abscess had Focal neurological deficit

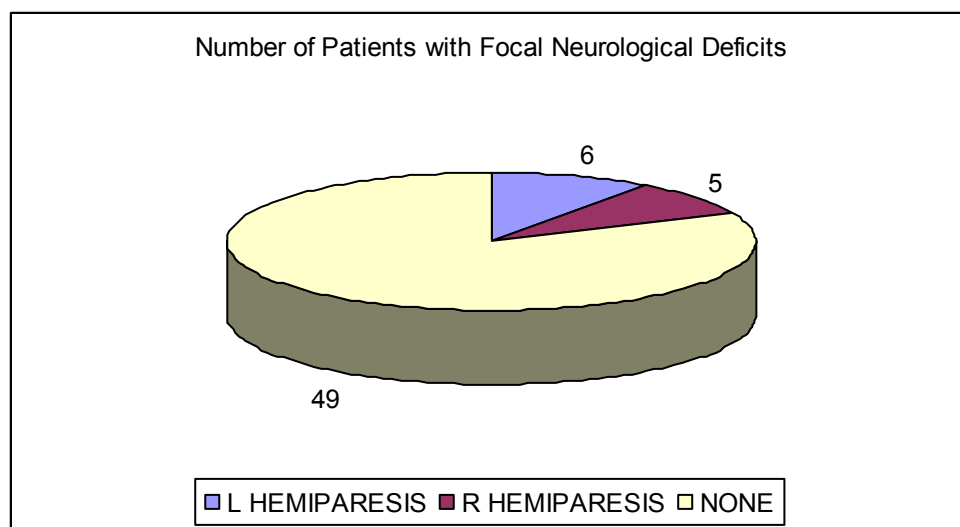


Chart 13: Clinical manifestation – Focal neurological deficit

#### 4f. Clinical manifestations – Others

OTHERS	FREQUENCY
ALTERED LEVEL OF CONSCIOUSNESS	13
ATAXIA	4
HAEMORRHAGIC DISORDER	1
JAUNDICE	1
Total	19

Table 14: Clinical manifestation – Others

19 of the 60 patients with brain abscess had other clinical manifestations

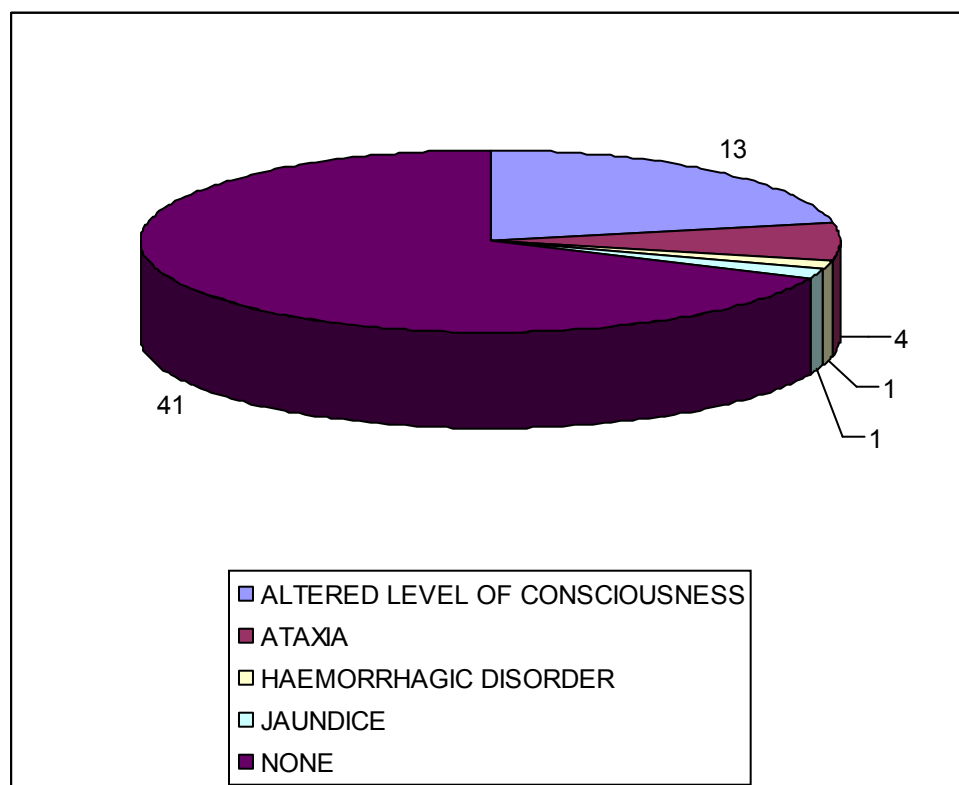


Chart 14: Clinical manifestation – Others



### 5a. Radiology – Side of Lesions

SIDE OF LESIONS	NUMBER OF PATIENTS
BIFRONTAL	2
LEFT	29
MULTIPLE	6
RIGHT	23
<b>TOTAL</b>	<b>60</b>

Table 15: Radiology – Side of Lesions

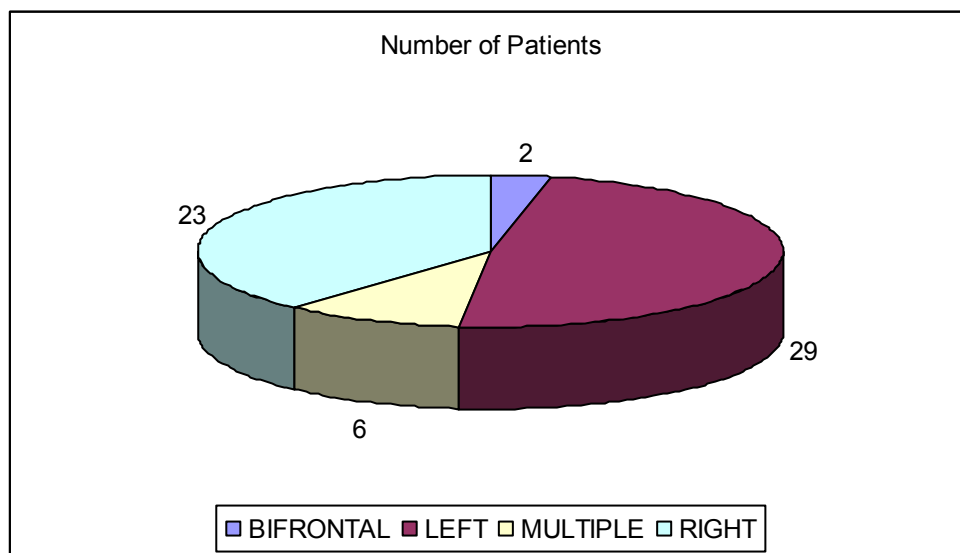


Chart 15: Radiology – Side of Lesions

## 5b. Radiology – site of lesion

LOCATION OF LESIONS	NUMBER OF PATIENTS
FRONTAL	13
TEMPORAL	8
PARIETAL	10
OCCIPITAL	3
GANGLIOCAPSULAR	2
CEREBELLUM	7
MULTIPLE	17
<b>TOTAL</b>	<b>60</b>

The location of abscess is shown in the above table.

Table 16: Radiology – site of lesion

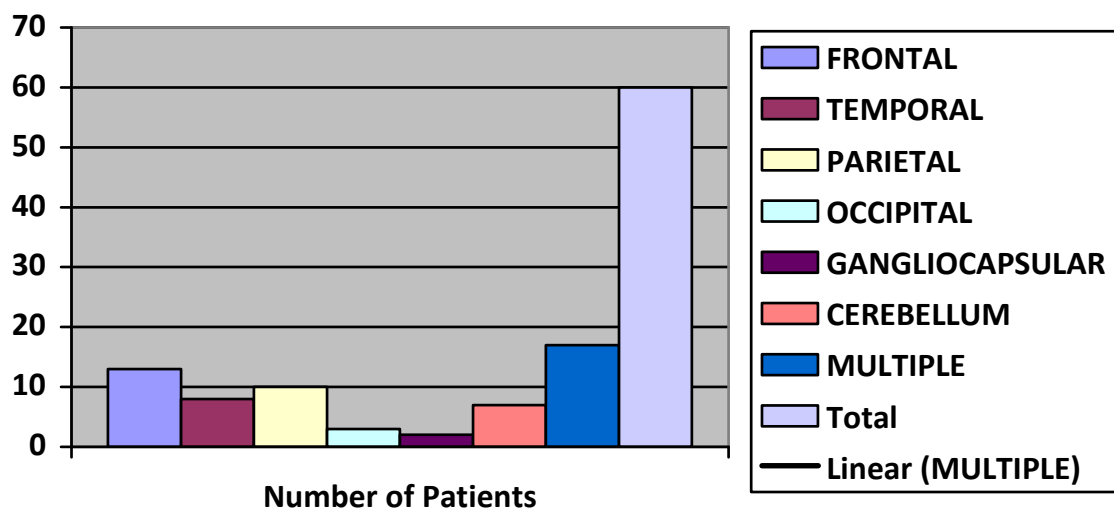


Chart 16: Radiology – site of lesion

## 6. Management

MANAGEMENT	NO. OF PATIENTS
AF TAPPING	1
BURR HOLE AND TAPPING	34
BURR HOLE & TAPPING + CRANIOTOMY AND EXCISION	2
BURR HOLE AND TAPPING + VP SHUNT RIGHT	1
CONSERVATIVE	8
CRANIOTOMY AND EXCISION	11
EVD RIGHT FRONTAL	1
STEREOTAXY	1
VP SHUNT RIGHT	1
<b>Total</b>	<b>60</b>

Table 17: Management of brain abscess

The abscesses were managed as shown in the above table

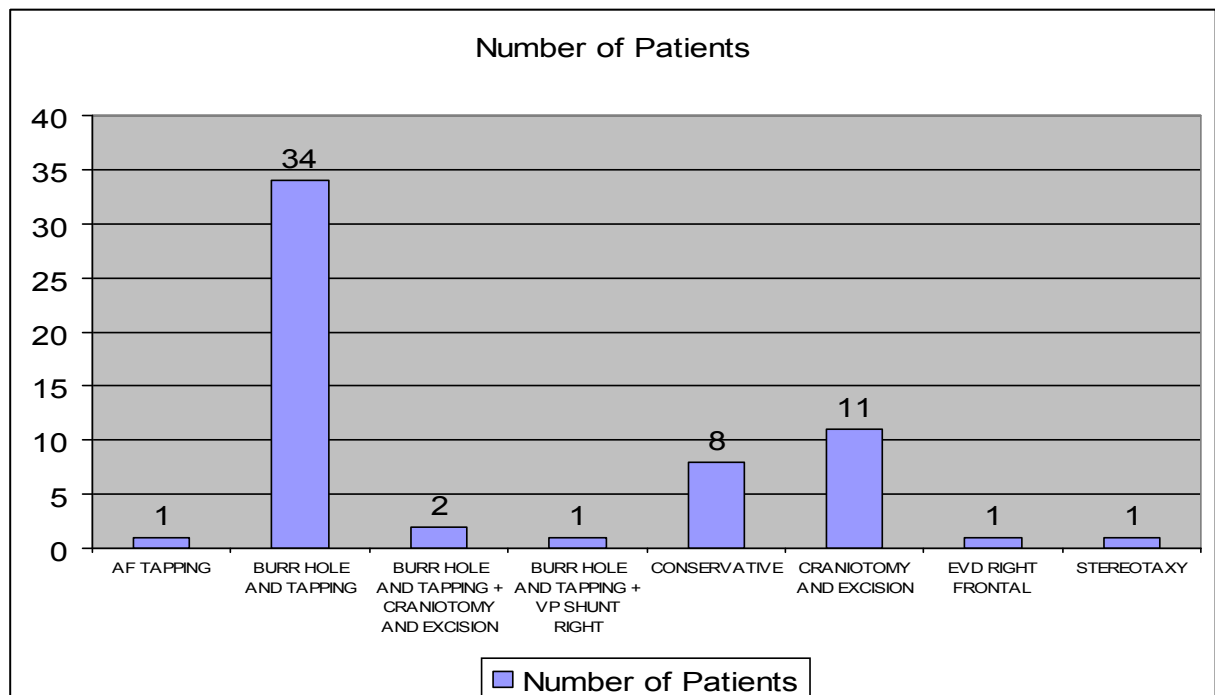


Chart 17: Management of brain abscess

## 6a. Management - Antibiotics

ANTIBIOTICS	NUMBER OF PATIENTS
AMPHOTERICIN B	1
ATT	4
ATT + CP, GM, METRO	1
CIPRO	3
CIPRO / AMIK	1
CIPRO + AMPHOTERICIN B	1
CIPRO / OFLOX	1
CO-TR	3
CO-TRIMAX / AMIKACIN	1
EMPIRICAL CP, GM, METRO	42
ERYTHRO	1
VANCOMY / CIPRO	1
<b>Total</b>	<b>60</b>

Table 19: Management – Antibiotics

As there was no growth in majority of the patients, empirical broad spectrum antibiotics were used in such patients.

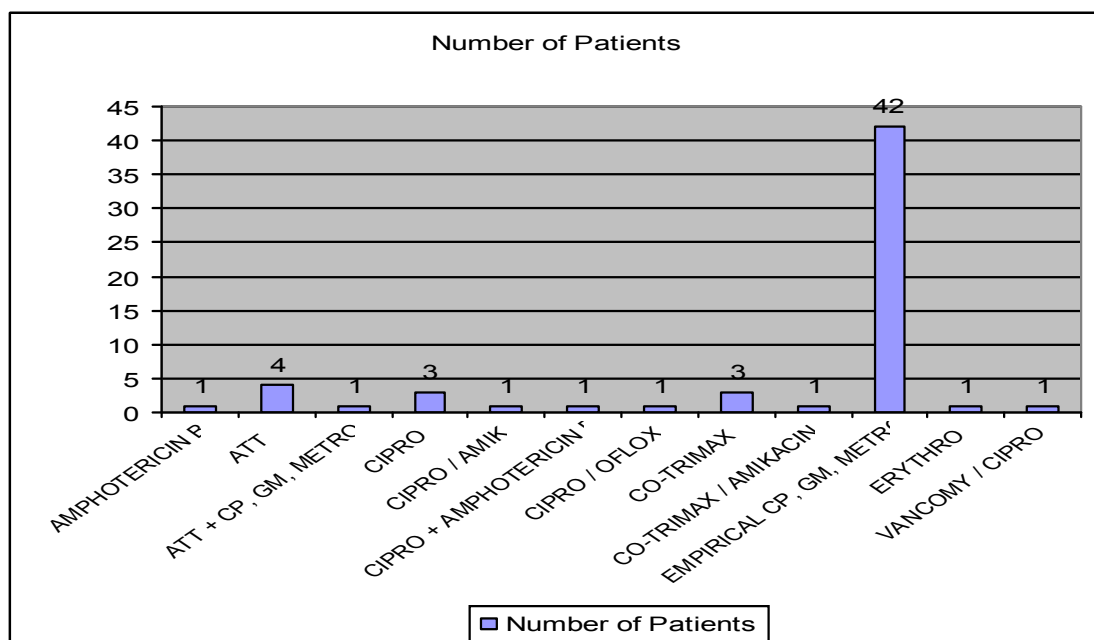


Chart 19: Management - Antibiotics

## 7. Microbiology

MICROBIOLOGY	NUMBER OF PATIENTS
AFB	2
ANAEROBES	1
STAPHYLOCOCCUS AUREUS	5
KLEBSIELLA	3
STREPTOCOCCUS	1
PSEUDOMONAS	1
FUNGI	1
ANAEROBES + FUNGAL	1
KLEBSIELLA + STAPHYLOCOCCUS	1
AFB + STAPH	1
NO GROWTH	35
<b>TOTAL</b>	<b>52</b>

Table 18: Microbiology of brain abscess

Of the total 60 patients 8 were managed conservatively. In the rest of 52 patients the organisms identified from pus were AFB, staphylococcus, pseudomonas, fungi and predominantly no growth was identified from pus in 35 patients.

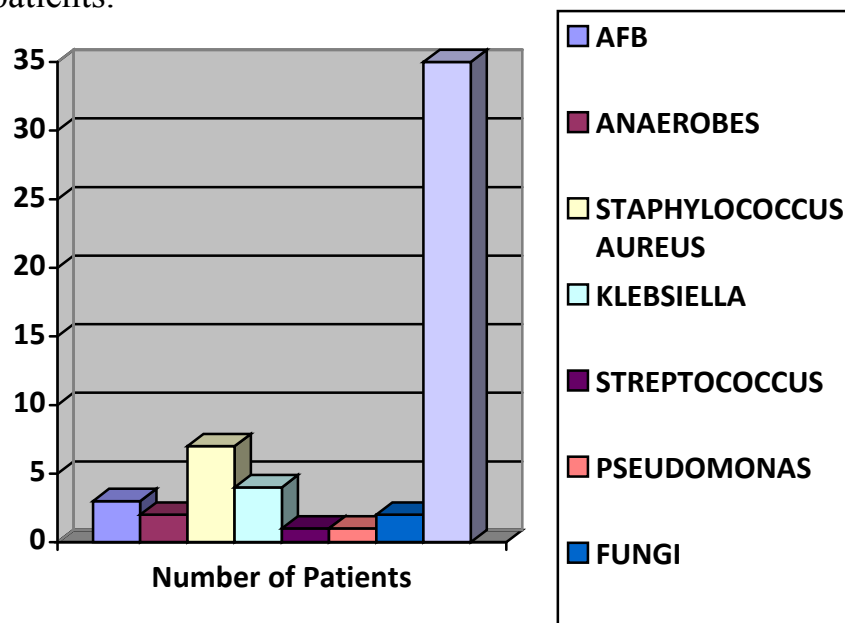


Chart 18: Microbiology of brain abscess

8a. Outcome

OUTCOME	NUMBER OF PATIENTS
DIED	7
IMPROVED	53
Total	60

Table 19: Outcome

In this study 7 patients died whereas 53 patients showed improvement.

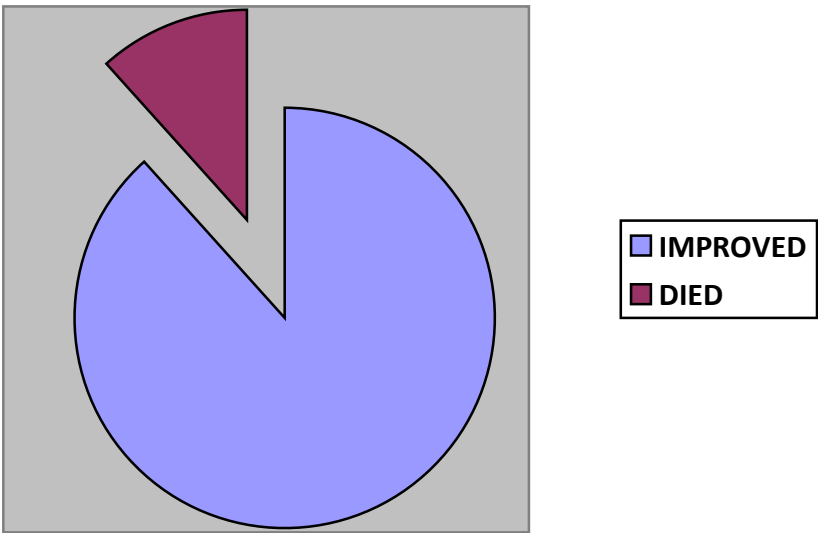


Chart 19: Outcome

**8b. AGE vs. OUTCOME:**

	<b>OUTCOME</b>		<b>Total</b>
<b>AGE</b>	<b>IMPROVED</b>	<b>DIED</b>	
Less than 1 year	11	2	13
1 to 12 years	6	2	8
13 to 20 years	11	1	12
21 to 30 years	9	2	11
31 to 40 years	8	0	8
41 to 50 years	3	0	3
51 to 60 years	3	0	3
61 to 70 years	2	0	2
<b>Total</b>	<b>53</b>	<b>7</b>	<b>60</b>

Chi square: 4.25      P=0.756 Not significant.

Table 20: AGE vs. OUTCOME

In this study 2 deaths occurred in less than one year age group, 2 deaths in 1-12 years, 1 death in 13-20 years and 2 deaths in 21 -30 years.

### 8c. SEX \* OUTCOME

		OUTCOME		Total
		DIED	IMPROVED	
SEX	FEMALE	2	14	16
	MALE	5	39	44
<b>Total</b>		<b>7</b>	<b>50</b>	<b>60</b>

P>0.05 Not significant

Table 21: SEX vs. OUTCOME

Among those dead 5 were males and 3 were females.

### 8d. Statistical analysis of heart disease and outcome

OUTCOME			
HEART DISEASE	DIED	IMPROVED	Total
TGAVSD	0	1	1
TOF	0	3	3
TOF + PULMONARY ATRESIA	1	0	1
	<b>7</b>	<b>53</b>	<b>60</b>

P>0.05 not significant

Only one patient with heart disease died among the 5 patients. Though the outcome appeared good it was not statistically significant.

Table 22: HEART \* OUTCOME



### 8e. ENT \* OUTCOME

ENT OUTCOME			
CONDITIONS	DIED	IMPROVED	Total
FRONTAL SINUSITIS	0	1	1
LEFT CSOM	1	5	6
RIGHT CSOM	0	3	3
	1	9	10

P>0.05 not significant

Table 23: ENT \* OUTCOME

The analysis revealed a P value >0.05 among the 10 patients with ENT causes.

### 8f. FEVER \* OUTCOME

FEVER OUTCOME		
DIED	IMPROVED	Total
6	46	52

Table 24: FEVER \* OUTCOME

6 patients died among the total of 52 patients who presented with fever.

### 8g. HEADACHE \* OUTCOME

HEADACHE OUTCOME		
DIED	IMPROVED	Total
4	34	38

P>0.05 Not significant

Table 25: HEADACHE \* OUTCOME

Analysing the outcome, 34 patients with headache had improved while 4 patients died.

### 8h. SEIZURES \* OUTCOME

SEIZURES	OUTCOME		
	DIED	IMPROVED	Total
	3	21	24

P>0.05 Not significant

Table 26: SEIZURES \* OUTCOME

Statistical analysis of seizure presentation revealed a P value >0.05

### 8i. VOMITING \* OUTCOME

OUTCOME			
VOMITING	DIED	IMPROVED	Total
NO VOMITING	3	21	24
VOMITING	4	32	36
<b>Total</b>	<b>7</b>	<b>53</b>	<b>60</b>

P>0.05 Not significant

Table 27: VOMITING \* OUTCOME

Among patients presenting with vomiting 4 had died.

### 8j. FOCAL NEUROLOGICAL DEFICITS \* OUTCOME

OUTCOME			
NEUROLOGICAL DEFICITS	DIED	IMPROVED	Total
L HEMIPARESIS	1	5	6
R HEMIPARESIS	1	4	5
<b>Total</b>	<b>7</b>	<b>53</b>	<b>60</b>

P>0.05 not significant

Table 28: FOCAL NEUROLOGICAL DEFICITS \* OUTCOME

Among the 11 patients with focal neurological deficits 2 patients died.

P>0.05

### 8k. SENSORIUM \* OUTCOME

OTHERS OUTCOME			
SENSORIUM	DIED	IMPROVED	Total
ALTERED LEVEL OF CONSCIOUSNESS	6	7	13
<b>Total</b>	<b>6</b>	<b>7</b>	<b>13</b>

P =0.0002 Significant

Altered level of consciousness was significant statistically.

Table 29: SENSORIUM \* OUTCOME

### 8l. CT BRAIN \* OUTCOME

LOCATION OF LESIONS	NUMBER OF PATIENTS		
	DIED	IMPROVED	TOTAL
FRONTAL	1	12	13
TEMPORAL	0	8	8
PARIETAL	2	8	10
OCCIPITAL	0	3	3
GANGLIOCAPSULAR	0	2	2
CEREBELLUM	0	7	7
MULTIPLE	4	13	17
<b>TOTAL</b>	<b>7</b>	<b>53</b>	<b>60</b>

### 8m. MANAGEMENT \* OUTCOME

		OUTCOME		TOTAL
		DIED	IMPROVED	
MANAGEMENT	AF TAPPING	0	1	1
	BURR HOLE AND TAPPING	6	28	34
	BURR HOLE & TAPPING + CRANIOTOMY & EXCISION	0	2	2
	BURR HOLE AND TAPPING + VP SHUNT RIGHT	0	1	1
	CONSERVATIVE	0	8	8
	CRANIOTOMY AND EXCISION	0	11	11
	EVD RIGHT FRONTAL	1	0	1
	STEREOTAXY	0	1	1
	VP SHUNT RIGHT	0	1	1
TOTAL		7	53	60

Table 31: MANAGEMENT \* OUTCOME

## 8n. MICROBIOLOGY \* OUTCOME

	IMPROVED	DEATH	TOTAL
NO GROWTH	29	6	35
CULTURE POSITIVE	16	1	17
<b>Total</b>	<b>45</b>	<b>7</b>	<b>52</b>

Table 32: MICROBIOLOGY \* OUTCOME

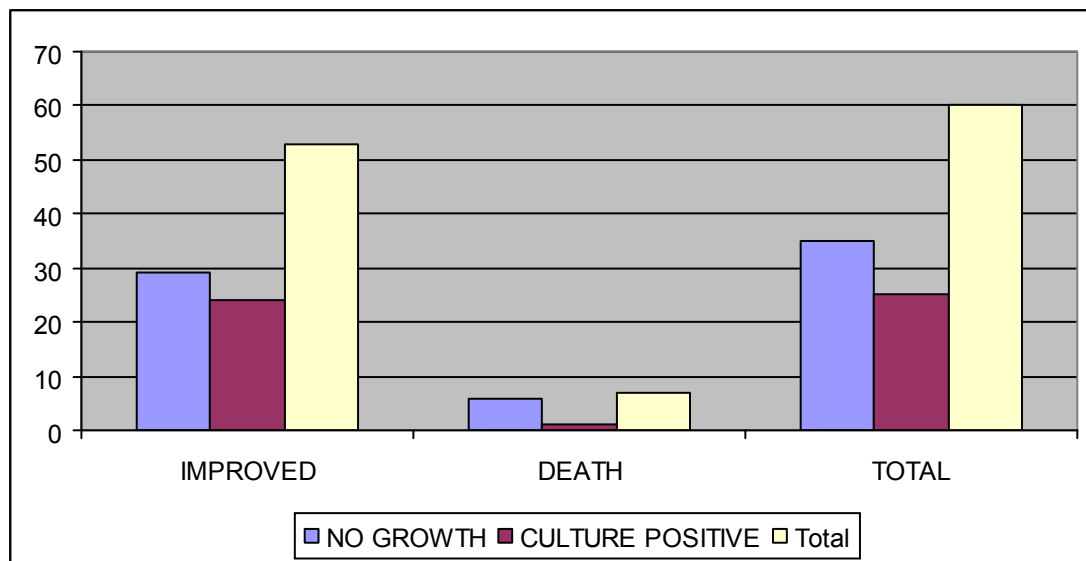


Chart 20: MICROBIOLOGY \* OUTCOME

## **ANALYSIS AND DISCUSSION**

## **ANALYSIS AND DISCUSSION**

60 patients were studied during the 3 year period, and an average of 20 cases per year and this study clearly reveals that brain abscess remains a significant neurosurgical problem even in this era of broad spectrum antimicrobial therapy.

### **1. Age**

It is well noticed that brain abscess occurs in any age. There is no age predilection and people of all age group are affected

According to McClelland et al, middle decade of life is common to this<sup>6, 33, 35</sup>, whereas Sinha et al stated that, patients below 20 years of age accounts for 74-89% of patients<sup>43</sup>. Though rare in neonates and infants, there are documented reports of brain abscess in this age group (Erdogon et al 2002)<sup>19</sup>. Most commonly patients belong to the age group of less than one year. Majority of cases occur between 2<sup>nd</sup> and 3<sup>rd</sup> decades of age. Majority of the patients in this study are below the age of 30 years.

This study revealed brain abscess is more common among patients less than one year and between 13 to 30 years. Deaths are also more in this age group probably due to less developed immune system under one year of age.



## **2. Sex**

When comparing the incidence of brain abscess, males outnumber the females in any age group<sup>9, 18,33,35,37</sup> except in the age group of less than one year. This predilection of male sex though unclear may be attributed to their occupation, road traffic accident etc common in male. Similar observation was reported by Lakshmi et al 1993<sup>29,30</sup>. Males outnumber females in this study.

## **3. Symptoms**

Though fever is an important symptom in the majority of cases, few cases do occur without fever<sup>10</sup>. As any other intracranial lesion which increases the intra cranial pressure and producing vomiting, brain abscess patients too have this symptom in majority of cases. The triad of fever, headache and vomiting is present in more than 50% of cases in this study. The conscious level at the time of admission is a reliable prognostic factor. According to Morgan et al 1973, Mampalam et al up to 2/3<sup>rd</sup> of patients had altered sensorium. 21% of patients in this study had altered sensorium. Majority of our cases > 80% is admitted with a GCS of >10. Cases with GCS <9 have a high mortality rate, which is reported in many series<sup>31, 35</sup>. This is well appreciated in this study also. Among the 60 patients, 52 had fever, 38 had headache, and 36 had vomiting. Seizures were present in 24 patients, hemiparesis in 11 cases and cerebellar

symptoms are present in 4 cases. The predisposing source causing brain abscess is present in 41 cases. CSOM is found to be the commonest source in 9 patients causing brain abscess. This is supported by a large series of studies from developing countries<sup>13</sup>. These otogenic abscesses are mainly solitary and located in either temporal lobe or in the cerebellum similar to the findings of (Carey et al 1972, Samson & Clark 1973)<sup>14</sup>.

### **Heart Diseases**

Cyanotic congenital heart disease (CHD) causing Brain abscess has been encountered in 5 cases. Brain abscess and cerebral thrombosis are the serious complications due to CHD. If the CHD is not corrected the micro infarcts caused by them become a nidus for the micro organisms resulting in bacteraemia causing abscess. As stated by Kagawa et al 1983<sup>26</sup>, Tetralogy of Fallot and transposition of great vessels are the major causes of brain abscess in this study also. In this study 3 are in the ages between 4-8 years, the other 2 are in 16-22 years, as reported by Fischer et al 1981, Kagawa et al 1983 peak age is between 4 to 8 years.

## **Road traffic accidents**

With the rise of road traffic accidents, industrialization terrorism etc incidence of head injury is increasing and more brain abscess occurs. In this study 6 cases 10% are due to trauma.

## **Infections**

Lung infections like bronchiectasis; pneumonia, pleural effusion was noted in 3 patients. Post craniotomy patients causing abscess is seen in 3 patients. The entry of micro organisms may be gained during the surgery. 4 patients had septic foci like gluteal abscess, pyelonephritis, skin pustules and cellulitis. Sinusitis is noted in 2 cases. Majority of the infants with brain abscess were found referred from paediatric ICU as neonatal sepsis. These infants are either pre term or small for gestation. These cases are more prone for infection, though the sources remain obscure.

## **Location**

In this study solitary lesions are more common, but multiple Lesions occurring simultaneously have also been reported. Haematogenous spread from distant source of infection because multiple abscesses. In multiple lesions each lesion is separated by a normal brain parenchyma. In multiloculated abscess no such normal parenchyma is seen between the lesions. Solitary lesions are present in 43 cases in our

study. The frontal lobe is involved in 13 cases, temporal lobe 8 cases, parietal lobe 10 cases, occipital lobe 3 cases and eloquent areas in 2 cases. In the cerebellum 6 patients had solitary lesions. Multiple lesions were present in 17 cases .

The eloquent areas like thalamus, gangliocapsular region were involved in 2 patients. Among these multiloculated abscesses were present in 9 patients. Symptoms of headache, fever and vomiting constitute an important triad in this study, which is present in > 60% of patients.

Depending on the predisposing factors certain lobes are more prone for brain abscess. In this study temporal lobe (4) and cerebellum (3) are more commonly involved in otogenic brain abscess. Similar observation was reported by Joshi 1988, Sinha et al 2003, Anand 2004. Parietal lobe was involved in a single patient with CSOM. In a single case of frontal lobe abscess frontal sinusitis is the predisposing factor.

### **Microbiology**

The pus aspirated from the abscess was subjected to microbiological examination like gram staining, acid fast bacilli staining and fungal staining. This helps in the Identification of micro organisms and by doing culture and sensitivity appropriate anti-microbial therapy

was initiated. The role of microbiologist is more vital for the tuberculous and fungal abscess, because distinguishing these by clinical and radiological methods is very difficult.

Culture was positive in 14 cases and 11 pus specimens yielded a single isolate. In the literature 44-100% of culture positivity was reported by (De Louvois et al 1977, Chandramukhi et al 1980, Sinha et al 2003, Lakshmi et al 1993, De et al 2000)<sup>15</sup>. 23% of culture positivity was observed in this study.

Aerobes are more common than anaerobes in this study. Similar results are reported in large series, but reports of anaerobes dominating aerobes are also present (Bhardwaj & John et al 1985)<sup>16</sup>. In this study Staphylococci and Klebsiella were more common organisms. Streptococci, Pseudomonas and anaerobes like Enterococci, bacteroides are also found. Fungal growth was present in two cases. Majority of the specimens do not have any growth. This may be attributed to the prior empirical, broad spectrum anti microbial therapy and the fastidious growth.

No organism was isolated in 9-63% of pus specimens as per Gregory et al 1967 series<sup>17</sup>. In our study the organisms are highly sensitive to third or fourth generation cephalosporins and vancomycin.

The anaerobes are sensitive to metronidazole. The fungal growth is sensitive to amphotericin-B and Itraconazole. No organism was isolated in 60% of pus specimens in this study.

## **Management**

The surgical procedures followed in this study were burr hole tapping, craniotomy and excision, stereotactic aspiration, external ventricular drainage and ventriculoperitoneal shunt. In Some patients combination of procedures has also been done.

For example in multiloculated and organized abscess repeated aspiration, followed by craniotomy was done. Here 34 cases undergone Burr hole tapping in which 13 cases were retapped.

Craniotomy and excision was done in 11 cases. Both aspiration and craniotomy and excision was done in 2 cases. Stereotactic aspiration was done in a single patient where the abscess was deep seated in the gangliocapsular region. In three cases where the abscess and the surrounding oedema caused CSF outflow obstruction two cases underwent v-p shunt and in the other neonate external ventricular drainage was done. The excised abscess were histopathologically examined and found to be non specific abscess.

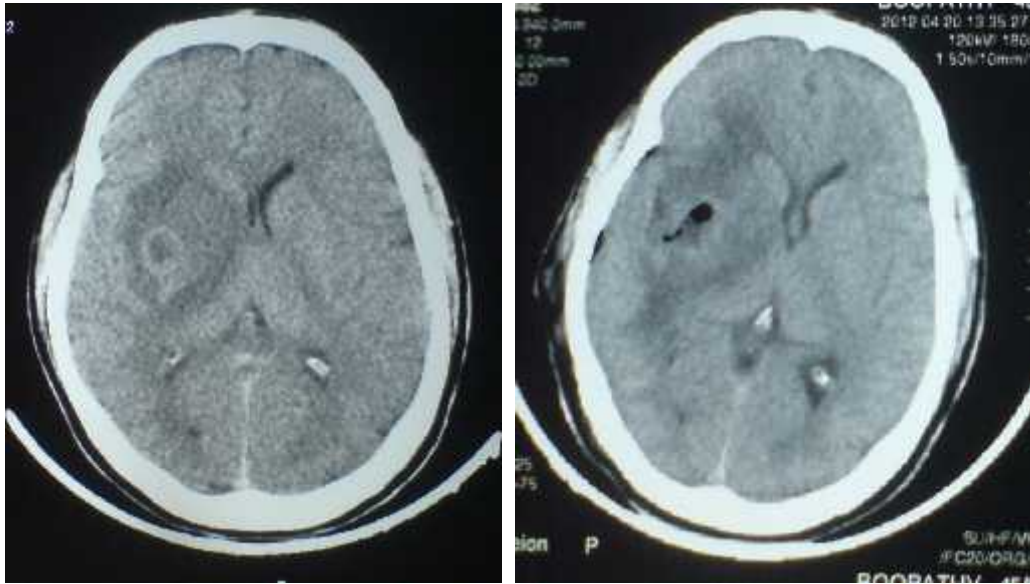


Image 1: Pre-operative and post-operative CT images of brain abscess – Stereotactic surgery



Image 2: CT images of a patient who developed post traumatic brain abscess – post trauma ct, pre & post-operative images

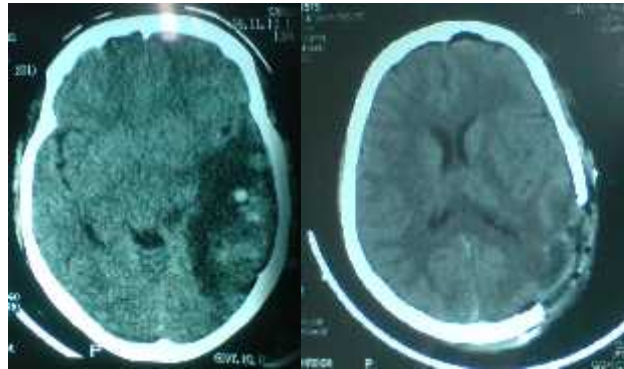


Image 3: Pre-operative and post-operative CT images of brain  
abscess non specific abscess

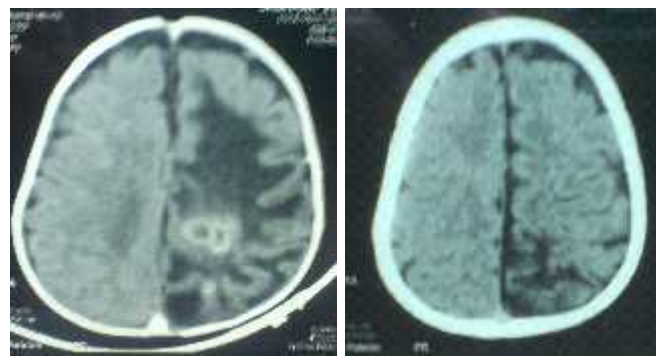


Image 4: Pre-operative and post-operative CT images of brain  
abscess burr hole tapping done.

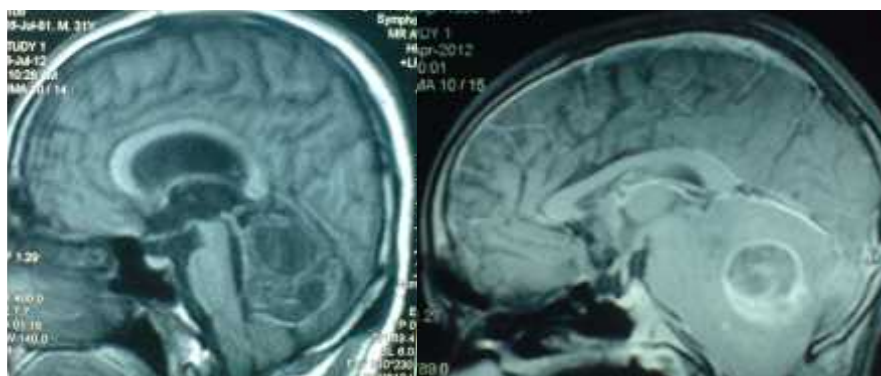


Image 5: Pre-operative images of cerebellar abscess due to  
otogenic causes.



## **Prognosis**

Among 60 cases, 53 patients improved and only seven patients expired, of which 4 were children and 3 were adults.

The age group ranges from 1 month to 30 years, four paediatric and three adult patients. In the paediatric cases two were neonates, the third one a 3 year old and the fourth one 7 year old with retro virus positive.

Staphylococcus was the predominant organism in this study.

The three adult patients expired were 16, 27 and 30 years of age. The 16 year old had TOF and CSOM. The 30 year old patient developed abscess due to post traumatic sequale. In common all these seven cases had altered sensorium as a common factor during admission. Similar observation was reported by Karamdam's & Shulman 1975 and McClelland et al 1978. My study showed that otogenic brain abscess had a better outcome than haematogenous type. Sinha et al had reported similar observation.

## **CONCLUSION**

## CONCLUSION

A study of 60 patients presenting with brain abscess has lead to the following conclusions.

1. Brain abscess still occurs in the modern era, better visualised with modern imaging studies.
2. Retro viral infections also contribute for brain abscess apart from other known causes, hence all cases of brain abscesses to be screened for retro viral infection
3. Neonates and children show poor prognosis and prompt aggressive management is essential for better outcome.
4. Constant vigil and surveillance will help for decreasing the magnitude of morbidity and mortality from brain abscess.

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## Ethical Committee

### INSTITUTIONAL ETHICS COMMITTEE MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301

Fax : 044 25363970

#### CERTIFICATE OF APPROVAL

To

Dr.T.Sureshababu,  
Post Graduate in Neurosurgery,  
Madras Medical College, Chennai -3

Dear Dr.T.Sureshababu,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "A Study on brain abscess" No.13122012.

The following members of Ethics Committee were present in the meeting held on 11.12.2012 conducted at Madras Medical College, Chennai -3.

- |  |                      |
|--|----------------------|
| 1. Dr.S.K.Rajan, M.D.FRCP, DSc   | --- Chairperson      |
| 2. Prof. R. Nandhini MD<br>Director, Instt. of Pharmacology ,MMC, Ch-3             | -- Member Secretary  |
| 3. Prof. Dr.A.Radhakrishnan MD<br>Director , Inst. Of Internal Medicine, MMC, Ch-3 | -- Member            |
| 4. Prof. Meenalochani, MD<br>Director , Instt. of O& G, Chennai                    | -- Member            |
| 5. Prof. Shyamraj MD<br>Director i/c , Instt. of Biochemistry , MMC, Ch-3          | -- Member            |
| 6. Prof. P. Karkuzhali. MD<br>Prof., Instt. of Pathology, MMC, Ch-3                | -- Member            |
| 7. Prof. S.Devivanayagam MS<br>Prof of Surgery, MMC, Ch-3                          | -- Member            |
| 8. Thiru. S. Govindsamy. BA, BL  | -- Lawyer            |
| 9. Tmt.Arnold Saulina MA MSW   | --- Social Scientist |

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

*R Nandini*  
Member Secretary, Ethics Committee

25/1/13

### INFORMED CONSENT FORM

Title of the study : A Study on Brain Abscess

Name of the Participant :

Name of the Institution :

Name and address of the sponsor/

agency (ies) (if any) :

#### Documentation of the informed consent

I \_\_\_\_\_ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in “ A Study on Brain Abscess ” (title of the study).

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past \_\_\_\_\_ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.
7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.
8. I have not participated in any research study within the past \_\_\_\_\_month(s).
9. I have not donated blood within the past \_\_\_\_\_ months—Add if the study involves extensive blood sampling.

9. I have not donated blood within the past \_\_\_\_\_ months—Add if the study involves extensive blood sampling.
10. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
11. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
12. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
13. I have understand that my identity will be kept confidential if my data are publicly presented
14. I have had my questions answered to my satisfaction.
15. I have decided to be in the research study. I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name : Signature : Date :

Name and Signature of impartial witness (required for illiterate patients):

Name : Signature : Date :

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name : Signature : Date :

For Children being enrolled in research:

Whether child's assent was asked : Yes / No (Tick one)

[If the answer to be above question is yes, write the following phrase:

You agree with the manner in which assent was asked for from your child and given by your child. You agree to have your child take part in this study].

[If answer to be above question No, give reason (s) : \_\_\_\_\_.

Although your child did not or could not give his or her assent, you agree to your child's participation in this study.

Name and Sign of/thumb impression of the participant's parent(s)(or legal representative)

Name : Signature : Date :

Name and Signature of impartial witness (required for parents of participant child illiterate):

Name : Signature : Date :

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent :

Name : Signature : Date :

**INSTITUTE OF NEUROLOGY**  
**GOVERNMENT GENERAL HOSPITAL, CHENNAI**

**PROFORMA**

Serial No:

Name:

Age:

MIN No:

I.P. No:

Sex: M / F

SOCIOECONOMIC STATUS

DOA

DOD

**HISTORY**

FEVER

HEADACHE

VOMITING

LEVEL OF CONSCIOUSNESS

FOCAL NEUROLOGICAL DEFICIT

**ASSOCIATED FEATURES**

CARDIAC DISEASE

ENT

SUPPURATIVE FOCUS

HIV/IMMUNODEFICIENCY

**INVESTIGATIONS**

CT BRAIN

LOBE PREFERENCE /

SINGLE /MULTIPLE

STAGE OF ABSCESS

MICROBIOLOGY

ORGANISM

CULTURE AND SENSITIVITY

**MANAGEMENT**

SURGERY

BURR HOLE / CRANIOTOMY /

OTHERS

CONSERVATIVE MANAGEMENT

STEREOTACTIC

**OUTCOME**

IMPROVED / DIED

## APPENDIX V –MASTER CHART

S.NO	AGE	SEX	FEVER	HEADACHE	VOMITING	SEIZURES	FOCAL NEUROLOGICAL DEFICITS	OTHERS	HEART	ENT	LUNG	TRAUMA	OTHER ASSOCIATED DISEASES	SIDE	CT BRAIN	MANAGEMENT	MICROBIOLOGY	HPE	ANTIBIOTICS	OUTCOME
1	24	F	+	+	+	+	RHP	-	NONE	NONE	NONE	-	NONE	LEFT	TEMPORAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
2	18	M	+	+	+	-	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	PARIETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
3	4	M	+	+	+	+	NFND	-	TOF	NONE	NONE	-	NONE	RIGHT	GANGLIOCAPS ULAR	CONSERVATIVE	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
4	30	M	+	+	+	-	NFND	ALOC	NONE	NONE	NONE	+	NONE	RIGHT	TEMPEROPARI ETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	EXP
5	14	F	+	-	-	-	NFND	-	NONE	RIGHT CSOM	NONE	-	NONE	RIGHT	CEREBELLUM	BURR HOLE AND TAPPING	BACTERIODS	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
6	36	M	+	+	-	-	NFND	-	NONE	RIGHT CSOM	NONE	-	NONE	RIGHT	TEMPORAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
7	17	M	+	+	+	-	NFND	JAU	NONE	NONE	NONE	-	NONE	RIGHT	FRONTAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCESS	EMPIRICAL CP, GM, METRO	DIS
8	35	M	+	+	-	-	RHP	-	NONE	LEFT CSOM	NONE	-	NONE	LEFT	TEMPORAL	BURR HOLE AND TAPPING	PSEUDOMONAS	NOT APPLICABLE	CIPRO	DIS
9	23	M	+	+	-	+	NFND	-	NONE	NONE	NONE	-	NONE	MULTIPLE	MULTIPLE	CONSERVATIVE	AFB	NOT APPLICABLE	CAT 1 ATT	DIS
10	40	M	+	-	-	-	NFND	ALOC	NONE	NONE	BRONCHIECTASIS	-	NONE	LEFT	OCCIPITAL	BURR HOLE AND TAPPING	KLEBSIELLA	NOT APPLICABLE	CIPRO	DIS
11	17	M	-	+	-	-	NFND	ATA	NONE	LEFT CSOM	NONE	-	NONE	LEFT	CEREBELLUM	BURR HOLE AND TAPPING	STREPTOCOCCI	NOT APPLICABLE	COTRI	DIS

12	2	F	+	-	-	+	LHP	-	NONE	NONE	NONE	-	NONE	RIGHT	PARIETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
13	51	M	+	+	-	-	NFND	ALOC	NONE	FRONTAL SINUSITIS	NONE	-	ORBITAL CELLULITIS	BIFRONTAL	BIFRONTAL	BURR HOLE AND TAPPING	ENTEROCOCCI, CANDIDA	NOT APPLICABLE	CIPRO, AMPHO B	DIS
14	15	F	+	+	+	-	LHP	-	NONE	NONE	NONE	+	NONE	RIGHT	FRONTAL	BURR HOLE AND TAPPING + CRANIOTOMY AND EXCISION	STAPHYLOCO CCUS AUREUS	NON SPECIFIC ABSCESS	SEPTRAN	DIS
15	30	F	-	-	-	+	NFND	-	NONE	NONE	NONE	-	NONE	RIGHT	FRONTAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCESS	EMPIRICAL CP, GM, METRO	DIS
16	20	M	+	+	+	+	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	TEMPORAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	ATT	DIS
17	16	M	+	+	+	+	NFND	-	NONE	NONE	NONE	-	PAPILLEDEMA	LEFT	CEREBELLUM	VP SHUNT RIGHT	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
18	0.25	F	+	-	-	+	NFND	HD	NONE	NONE	NONE	-	NONE	LEFT	FRONTOPARIE TAL	BURR HOLE AND TAPPING	KLEBSIELLA	NOT APPLICABLE	CIPRO	DIS
19	7	M	-	-	-	-	LHP	ALOC	NONE	NONE	NONE	-	HIV, PAPILLEDEMA, ALTERED CONCIOUSNESS	RIGHT	FRONTOPARIE TAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	EXP
20	0.33	M	+	-	-	+	NFND	-	NONE	NONE	NONE	-	NONE	RIGHT	TEMPORAL	CRANIOTOMY AND EXCISION	STAPHYLOCO CCUS AUREUS	NON SPECIFIC ABSCESS	ERYTHRO	DIS
21	45	M	+	+	-	+	LHP	-	NONE	NONE	NONE	-	NONE	LEFT	GANGLIOCAPS ULAR	STEREOTAXY	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
22	22	F	-	-	-	+	RHP	ALOC	TOF	NONE	NONE	-	NONE	LEFT	PARIETAL	CONSERVATIVE	AFB	NOT APPLICABLE	CAT 2 ATT	DIS



23	14	M	+	+	+	-	NFND	-	NONE	LEFT CSOM	NONE	-	NONE	LEFT	CEREBELLUM	BURR HOLE AND TAPPING + VP SHUNT	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
24	28	M	+	+	-	-	NFND	ATA	NONE	LEFT CSOM	NONE	-	NONE	LEFT	CEREBELLUM	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
25	60	M	+	+	+	-	NFND	-	NONE	NONE	NONE	-	NONE	MULTIPLE	MULTIPLE	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
26	16	M	+	+	+	-	NFND	ALOC	TOF + PULMONARY ATRESIA	NONE	NONE	-	NONE	RIGHT	PARIETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	EXP
27	15	M	+	+	-	-	NFND	ATA	NONE	LEFT CSOM	NONE	-	NONE	LEFT	CEREBELLUM	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	EXP
28	48	M	+	+	+	+	NFND	-	NONE	NONE	NONE	+	NONE	RIGHT	FRONTAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
29	40	M	+	+	+	-	NFND	-	NONE	NONE	NONE	-	NONE	RIGHT	TEMPEROCC IPITAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
30	18	M	+	+	+	-	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	FRONTAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCESS	EMPIRICAL CP, GM, METRO	DIS
31	35	M	+	+	+	-	NFND	ALOC	NONE	NONE	NONE	-	NONE	BIFRONTAL	BIFRONTAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCESS	EMPIRICAL CP, GM, METRO	DIS
32	67	M	+	+	+	-	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	PARIETAL	BURR HOLE AND TAPPING	NOCARDIA	NOT APPLICABLE	AMPHOTERICI N	DIS
33	0.33	F	+	-	+	-	NFND	-	NONE	NONE	NONE	-	Pre term / SEPSIS	LEFT	TEMPORAL	CONSERVATIV E	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS

34	5	M	+	+	+	-	NFND	ALOC	NONE	NONE	NONE	-	NONE	LEFT	PARIETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
35	7	M	+	+	+	-	NFND	-	TOF	NONE	NONE	-	NONE	RIGHT	PARIETOOCIPITAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
36	0.083	M	+	-	-	-	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	FRONTAL	CONSERVATIVE	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
37	0.41	M	+	-	+	+	NFND	-	NONE	NONE	NONE	-	NONE	RIGHT	FRONTAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
38	1	M	+	-	+	+	NFND	-	NONE	NONE	NONE	-	NONE	RIGHT	TEMPORAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCESS	EMPIRICAL CP, GM, METRO	DIS
39	0.41	F	+	-	+	+	NFND	-	NONE	NONE	PNEUMONIA	-	NONE	LEFT	PARIETAL	CONSERVATIVE	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
40	21	M	+	+	+	+	NFND	-	NONE	NONE	PNEUMONIA-SLE	-	NONE	MULTIPLE	MULTIPLE	CONSERVATIVE	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
41	14	F	+	+	+	+	NFND	-	NONE	NONE	NONE	-	HIV	LEFT	PARIETAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCESS	EMPIRICAL CP, GM, METRO	DIS
42	27	M	+	+	-	+	NFND	ALOC	NONE	NONE	NONE	-	NONE	LEFT	FRONTAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	EXP
43	3	F	+	-	+	+	RHP	ALOC	NONE	NONE	NONE	-	NONE	LEFT	FRONTOPARIETAL	BURR HOLE AND TAPPING	STAPHYLOCOCCUS AUREUS	NOT APPLICABLE	CO-TRIMAX,AMIK	EXP
44	8	M	-	+	+	+	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	PARIETOOCIPITAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	ATT	DIS
45	0.083	F	+	-	+	+	NFND	-	NONE	NONE	NONE	-	SEPSIS	MULTIPLE	MULTIPLE	BURR HOLE AND TAPPING	KLEBSIELLA, STAPHYLOCOCCUS AUREUS	NOT APPLICABLE	VANCOMY,CIPRO	DIS

46	49	M	-	-	-	-	RHP	ALOC	NONE	NONE	NONE	+	NONE	LEFT	PARIETAL	AFB + CRANIOTOMY AND EXCISION	STAPHYLOCO CCUS AUREUS	NON SPECIFIC ABSCCESS	ATT CP, GM, METRO	DIS
47	0.083	F	+	-	+	+	NFND	ALOC	NONE	NONE	NONE	-	Pre term / SEPSIS	RIGHT	OCCIPITAL	EVD RIGHT FRONTAL	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	EXP
48	0.083	F	+	-	-	-	NFND	-	NONE	NONE	NONE	-	Pre term / SEPSIS	LEFT	FRONTAL	BURR HOLE AND TAPPING	KLEBSIELLA	NOT APPLICABLE	CIPRO,OFLOX	DIS
49	0.083	M	+	-	+	+	NFND	-	NONE	NONE	NONE	-	Pre term / SEPSIS	RIGHT	OCCIPITAL	BURR HOLE AND TAPPING	STAPHYLOCO CCUS AUREUS	NOT APPLICABLE	SEPTRAN	DIS
50	35	M	+	+	+	-	NFND	ATA	NONE	RIGHT CSOM	NONE	-	NONE	RIGHT	CEREBELLUM	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
51	8	M	+	+	+	-	NFND	-	TGAVSD	NONE	NONE	-	NONE	RIGHT	TEMPEROPARI ETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
52	0.166	F	+	-	+	-	NFND	-	NONE	NONE	NONE	-	NONE	MULTIPLE	MULTIPLE	AF TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
53	39	M	+	+	+	+	NFND	-	NONE	LEFT CSOM	NONE	-	NONE	LEFT	TEMPORAL	BURR HOLE AND TAPPING	Coagulase Negative STAPHYLOCO CCUS AUREUS	NOT APPLICABLE	CIPRO, AMIK	DIS
54	25	M	+	-	-	-	LHP	-	NONE	NONE	NONE	+	NONE	RIGHT	FRONTOPARIE TAL	BURR HOLE AND TAPPING + CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCCESS	EMPIRICAL CP, GM, METRO	DIS
55	58	M	-	+	-	+	NFND	-	NONE	NONE	NONE	-	NONE	RIGHT	FRONTAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCCESS	EMPIRICAL CP, GM, METRO	DIS

56	24	M	-	+	-	-	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	PARIETAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
57	25	M	+	+	+	-	NFND	-	NONE	NONE	NONE	+	NONE	LEFT	FRONTAL	BURR HOLE AND TAPPING	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS
58	63	M	+	+	+	-	LHP	ALOC	NONE	NONE	NONE	-	NONE	RIGHT	TEMPEROPARI ETAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCCESS	EMPIRICAL CP, GM, METRO	DIS
59	38	F	+	+	+	+	NFND	-	NONE	NONE	NONE	-	NONE	LEFT	TEMPEROPARI ETAL	CRANIOTOMY AND EXCISION	NO GROWTH	NON SPECIFIC ABSCCESS	EMPIRICAL CP, GM, METRO	DIS
60	0.5	M	+	-	+	-	NFND	-	NONE	NONE	NONE	-	THIGH ABSCCESS ,CGD	MULTIPLE	MULTIPLE	CONSERVATIV E	NO GROWTH	NOT APPLICABLE	EMPIRICAL CP, GM, METRO	DIS

## **ABBREVIATIONS USED**

M = MALE

F = FEMALE

+ = PRESENT

- = ABSENT

NFND = NO FOCAL NEUROLOGICAL DEFICIT

RHP = RIGHT HEMIPARESIS

LHP = LEFT HEMIPARESIS

ALOC = ALTERED LEVEL OF CONSIOUNESS

JAU = JAUNDICE

ATA = ATAXIA

HD = HAEMORRHAGIC DISEASE

DIS = DISCHARGE

EXP = EXPIRED

HIV = HUMAN IMMUNODEFICIENCY VIRUS

SOL = SPACE OCCUPYING LESION

## **APPENDIX VI – TURNITIN REPORT FOR PLAGIARISM**



## Turnitin Originality Report

~~a study on brain abscess~~ by Sureshbabu  
Thirumal 18101512 M.Ch. Neuro Surgery  
From Medical (TNMGRMU APRIL 2013  
EXAMINATIONS)

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[S. Menon. "Current epidemiology of intracranial abscesses: a prospective 5 year study", Journal of Medical Microbiology, 10/01/2008](#)
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